Electrocardiogram 2: interpretation and signs of heart disease

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Abstract An electrocardiogram assesses the heart’s electrical activity. It is commonly used as a non-invasive monitoring device in many different healthcare settings. This article, the second in a three-part series, focuses on interpretation and the importance of being able to quickly identify key signs of myocardial infarction.


In this article...

- The six steps to interpreting a 12-lead electrocardiogram (ECG)
- What different wave patterns mean for potential diagnosis
- Common ECG changes in myocardial infarction

Key points

Electrocardiograms are investigations that assess the electrical activity of the heart.

There are six initial steps to take for the interpretation of a 12-lead electrocardiogram.

Other features to assess include the ST segment, T-wave and QT interval.

Clear documentation should include: the time monitoring started and ended; indications for monitoring; and significant patient events during the process, such as chest pain.

It is important to be able to quickly spot key signs of myocardial infarction.

ECG parameters

As covered in part 1, an ECG is a non-invasive method of monitoring the electrical activity of the heart. It is recorded onto specialised ECG paper, which runs at 25mm/second; the vertical (y) axis of the ECG shows voltage, while time is represented on the horizontal (x) axis.

ECG interpretation

When interpreting an ECG trace, the first thing to consider is the clinical history, for example, a history of chest pain or palpitations, which may be the reason for using a 12-lead ECG. It is also important to make a note of any key drugs that may affect rhythm or heart rate (such as beta-blockers or digoxin) and consider any abnormal blood test results – such as high or low potassium, magnesium or calcium levels – that may have an impact on the heart.

A stepwise approach should be taken to ECG rhythm interpretation. The first step should always be to confirm the patient’s details (name, date of birth, hospital/NHS number) and document this on the trace. After this, a six-stage approach to interpreting the ECG – outlined in Box 1 – should be used, as suggested by the Resuscitation Council UK.

Box 1. The six stages of ECG rhythm interpretation

1. Is there any electrical activity?
2. What is the ventricular (QRS) rate?
3. Is the QRS rhythm regular or irregular?
4. Is the width of the QRS complex narrow or broad?
5. Is atrial activity present?
6. Is atrial activity related to ventricular activity and, if so, how?

Source: Bit.ly/ResusUKECG

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Figure 1. Measuring heart rate using the R-R interval

Stage 1: does the ECG show electrical activity?
After checking the patient clinically at the bedside for airway, breathing and circulation, you should next confirm that electrical activity is being recorded from a cardiac monitor. It is important to check that the quality of the ECG recording looks appropriate, including having a stable baseline and no artefact. For a 12-lead ECG, it is crucial to ensure correct placement of the limb leads (I, II, III, avF, avL, avR) and chest leads (V1-V6) in the first instance (see part 1).

Stage 2: what is the ventricular rate?
After confirming that there is electrical activity, now consider the ventricular (or heart) rate from the ECG trace. The normal heart rate is 60-100 beats/minute (bpm); a heart rate of <60bmp is referred to as bradycardia, while one of >100bmp is referred to as tachycardia.

As described in part 1, an ECG complex consisting of the P-QRS-T wave components (P = atrial depolarisation; QRS = ventricular depolarisation; T = ventricular repolarisation) represents a cardiac cycle. The heart rate can be calculated by looking at the R-R interval between two consecutive QRS complexes. On standard pink ECG paper, each large square is made up of five small mm squares; each small square corresponds to 0.04 seconds (s) or 40 milliseconds (ms) and each large square is 0.20s (200ms). The R-R interval can be calculated by dividing 300 by the number of large squares between the R-waves in consecutive QRS complexes. For example, to calculate the heart rate in the ECG shown in Fig 1, 300 is divided by four, giving a heart rate of 75bpm.

Stage 3: is the QRS rhythm regular?
If there is a regular rhythm, there will be the same number of squares between consecutive QRS complexes. Sinus rhythm, bradycardia and tachycardia are all regular rhythms. One easy way to look at rhythm uses a paper recording from a 12-lead ECG. Looking at the long rhythm strip representing lead II, overlap a piece of paper and mark out each QRS complex. If the rhythm is regular, this marked paper will match the QRS complexes on any part of the rhythm strip.

Regularity can be difficult to detect in some tachyarrhythmias due to the fast heart rate and, sometimes, when the patient is given medications to slow the heart rate, the true rhythm becomes apparent. This will be discussed more in part 3.

Stage 4: what is the width of the QRS complex?
The QRS width is assessed by measuring the number of small squares between the beginning of the Q-wave and the end of the S-wave. This duration should be less than three small squares (<0.12s). An interval of >0.12s is a broad QRS complex, which suggests a rhythm that originates from the ventricles or a normal rhythm with a block in conduction of impulses from above the ventricles (such as right or left bundle branch block).

In contrast, a narrow QRS complex of <0.12s suggests that cardiac rhythm originates in the atria or the atrioventricular junction (above the ventricles). This means that assessing the QRS complex in the context of tachycardia can help differentiate a narrow or broad complex tachycardia, which suggest a supraventricular or ventricular tachycardia, respectively.

The QRS complex also represents how conduction is travelling down the ventricles via the right and left bundles, which originate from the bundle of His, as described in part 1. If there is a conduction block to the bundle branches, this may manifest with a widened QRS complex duration. Further analysis can help differentiate on which side (right or left) this occurs and will be discussed in more detail in part 3, which focuses on arrhythmias.

Stage 5: is there atrial activity?
It is important to check whether the patient is in sinus rhythm — that is, the rhythm is originating in the sinoatrial node, which is represented by the presence of normal P-waves. When reviewing an ECG, consider whether P-waves are present and followed by a QRS complex. P-waves can be assessed for shape, for example:

- Tall – seen in right atrial enlargement or low potassium states;
- Bifid (‘M’-shaped) – observed in left atrial enlargement;
- Saw-tooth – seen in atrial flutter.
If there are no P-waves, the patient is not in sinus rhythm. A common reason for this is atrial fibrillation, in which normal electrical activity in the atria (P-wave) is replaced by chaotic electrical signals.

Stage 6: is atrial activity related to ventricular activity?
To assess whether atrial activity is related to ventricular activity requires consideration of the time interval between the P-wave and QRS complex. This is called the PR interval and is normally 0.12-0.20s (represented by 3-5 small squares). The PR interval should be calculated and consistency assessed throughout the rhythm strip. When there is an abnormally short PR interval, this may suggest the P-wave is not originating in the sinoatrial node; the origins are perhaps closer to the atrioventricular (AV) node so the conduction takes less time. A short PR may also be due to an accessory electrical pathway that acts as a shortcut between the atria and ventricles. A more prolonged PR interval (>0.20s, or more than five small squares) may suggest a delay in the transmission of the atrial impulse to the ventricles; this is referred to as AV block. Details of the types of AV block will be covered in part 3.

Other considerations
When interpreting a 12-lead ECG, the six-step method described can be used as a starting point. Other features can also be assessed, as summarised in Box 2. A 12-lead ECG will provide information as described earlier, but can also be used to assess the cardiac axis, which is the direction of electrical impulse transmission across the heart. A left- or right-axis deviation can sometimes occur in a normal heart but may also be a useful indication of disease. For example, a right-axis deviation may be observed in cases of right ventricular hypertrophy, pulmonary embolism and
myocardial infarction; left-axis deviation may occur as part of a bundle branch block (discussed later) or myocardial infarction. Looking at leads I, II and III will help determine this, as shown in Fig 2.

**ST segment**
The ST segment is the part of the ECG between the end of the S-wave and the start of the T-wave. In a healthy individual, it should not be elevated or depressed, and is referred to as isoelectric. Abnormalities of the ST segment should be investigated to rule out problems such as ischaemic heart disease or pericarditis (inflammation of the pericardium (lining of the heart)).

**T-wave**
A tall T-wave may suggest electrolyte abnormalities such as hyperkalaemia, while an inverted wave may be a sign of – among other conditions – ischaemia, pulmonary embolism and left-ventricular hypertrophy.

**QT interval**
The QT interval is the time duration between the onset of the QRS complex and the end of the T-wave; it is calculated using lead II or chest leads V5-6. A short or prolonged QT interval can indicate underlying cardiac disorders, such as ischaemia, an underlying genetic cause of prolonged QT interval, and systemic disorders, such as an electrolyte imbalance (typically low potassium or low magnesium).

A prolonged QT may be caused by certain medications, which include antiarrhythmic drugs, antihistamines, antipsychotics, and antimalarials, as well as certain antibiotics such as erythromycin and clarithromycin (van Noord et al, 2010). It is important that nurses can recognise life-threatening arrhythmias, and have received immediate (Bit.ly/ResusUKILS) and advanced life-support training. More information and guidance on this has been published by the Resuscitation Council (2021) and the Society for Cardiological Science and Technology (2020).

**Documentation**
After assessing the ECG, it is important to ensure your findings are appropriately documented. A simple approach to ECG documentation is shown in Box 3.

**Ischaemic heart disease**
This next part will focus on identifying ischaemia changes in ECGs, as ischaemic (coronary) heart disease is the most common cause of death in the UK (National Institute for Health and Care Excellence, 2018). Rapid identification of patients presenting with acute signs of myocardial ischaemia or infarction is crucial, as patients require timely care and intervention to preserve their myocardium and prevent death. For most patients this will mean initial management, with pain relief and aspirin, with the potential for additional antiplatelet drugs to boost their chances of survival (Baigent et al, 1998).

Patients with marked ECG changes suggesting an acute myocardial infarction will require interventional procedures in a short window of effectiveness (Jarvis and Saman, 2017).

**ECG interpretation in myocardial ischaemia and infarction**
Ischaemic heart disease affecting the coronary arteries supplying the myocardium may lead to a myocardial infarction. There are key branches of the major coronary arteries that supply the heart and potential abruption to the supply in a particular artery will show a typical regional pattern of abnormal ECG findings (Table 1). It is important that health professionals are able to recognise these.

The universal definition of myocardial infarction includes raised cardiac biomarkers (notably cardiac troponin released due to myocardial injury), with ≥1 value above the 99th percentile of the upper reference limit and/or a rise in cardiac biomarkers with ≥1 value of the features below (Thygesen et al, 2012):

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**Box 2. Checklist: 12-lead ECG interpretation**
When interpreting a 12-lead ECG, check:
- Rate (60-100 beats per minute)
- Rhythm
- P-waves
- PR interval (0.12-0.2 seconds)
- QRS complex (<0.12 seconds)
- QT interval (<0.44 seconds)
- ST segment
- T-waves
- Cardiac axis

**Box 3. ECG documentation checklist**
- Demographics, including full name, date of birth and unique patient identifier
- Time monitoring started and finished
- Indications for monitoring
- Any significant patient events during monitoring, for example, chest pain, palpitations, dizziness, dyspnoea

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**Fig 2. Assessing the cardiac axis**

- Normal cardiac axis
- Right axis deviation
- Left axis deviation
Table 1. Abnormal ECG readings in myocardial infarction

<table>
<thead>
<tr>
<th>Myocardial infarction type</th>
<th>ECG component</th>
<th>Region of heart supplied by artery</th>
<th>ECG changes typically ST elevation+/- Q-waves</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inferior</td>
<td>Left anterior descending artery - from left coronary artery</td>
<td>Anterior wall of left ventricle, anterior septum, bundle branches</td>
<td>Leads II, III, aVF</td>
</tr>
<tr>
<td>Anterolateral</td>
<td>Left circumflex - from left coronary artery</td>
<td>Left lateral wall (atrium, left ventricle), posterior wall left ventricle</td>
<td>Leads I, aVL, V5, V6</td>
</tr>
<tr>
<td>Anterior</td>
<td>Right coronary artery</td>
<td>Right atrium, right ventricle, inferior part of the left ventricle, posterior septum</td>
<td>Leads VI-V6</td>
</tr>
<tr>
<td>Posterior</td>
<td>Right coronary artery (branch)</td>
<td>Posterior heart</td>
<td>ST depression in V1-V4. ST elevation if posterior leads placed on back.</td>
</tr>
</tbody>
</table>

Fig 3. ECG showing T-wave inversion in NSTEMI

- Symptoms of cardiac ischaemia;
- New ECG changes indicating new ischaemia (for example, new ST segment or T-wave changes or new left-bundle branch block);
- Development of pathological Q-wave in the ECG;
- Evidence of new loss of viable myocardium or new regional wall motion abnormality on imaging, such as echocardiography.

There are specific ECG changes in the ST segment that help to diagnose an ST elevation myocardial infarction (STEMI) compared with a non-STEMI. In a STEMI, there is ST elevation of ≥1mm in two adjacent chest leads or two mm ST elevation in two adjacent limb leads, or development of the new left-bundle branch block. These ECG changes in a patient with a history of chest pain indicate the need for urgent cardiology review.

The decision to be made is whether the patient will need immediate reperfusion therapy, such as primary percutaneous coronary intervention (PCI) using coronary angioplasty (with or without stent insertion) to restore flow in the affected coronary artery or arteries. Sometimes, thrombolyis drugs, such as intravenous alteplase, may be used, although PCI is favoured. In a patient presenting with non-STEMI, there may be ischaemic changes such as ST depression or T-wave inversion (Fig 3), accompanied by a rise in the cardiac troponins. These findings indicate the patient may need inpatient angiography along with antiplatelet agents and possibly a beta-blocker (Collet et al, 2020; Valgimiglì et al, 2018).

Conclusion
Understanding the approach to interpreting an ECG, and at the very least being able to recognise common pathology, is an important skill. Ischaemic heart disease is a common cause of mortality and recognition of the ECG signs of a STEMI are important as rapid decisions have to be made about treatment. Part 3 will focus on disorders in cardiac rhythm and understanding and recognising life-threatening arrhythmias, as well as conduction defects, their causes and management. NT